which is the special feature in the pathology of rickets. The epiphysal lines of cartilage, by the growth of which the shafts of the long bones are lengthened, are not formed in undue quantity, as has been stated, but, in consequence of the imperfection of calcification, they exist in undue proportion, and, yielding under pressure, they bulge or are squeezed out, and, becoming subsequently ossified, form the circular projecting ridges so prominent in this and some other specimens. This condition, which is sometimes thought to be a result of rheumatic arthritis, but has no relation to that disease, is most common and most marked at the lower end of the thigh bone, because the epiphysal line here is the one in which growth takes place longer and at a greater rate than in any other part of the skeleton, and in which, therefore, defect in the growing processes, and especially in the ossification of the growing line of cartilage, is most likely to take place. Indeed, it is not infrequently the only part in which this bulging is observed in the adult skeleton; and, occasionally, the bulging here is the only persistent feature of rickets. I say in the adult skeleton, for it is curious and rather difficult of explanation that, although the bulging of the lower epiphysal line of the radius and the sternal ends of the ribs is so often found in rickety children, this feature commonly disappears as the children grow up, and it is seldom found in any marked degree in rickety skeletons in any part except the lower end of the thigh bone and the upper end of the arm bone.

It is mainly owing to this deficiency of cartilage growth and cartilage ossification at the epiphysal lines that rickety persons are usually short-limbed, their limbs not growing up to a due proportion with the head and trunk. This is especially the case with the lower limbs, and, above all, with the thighs. In many rickety skeletons, the thigh bones are not so long as the leg bones, and the expressed epiphysal lines are then, as in this specimen, very marked. In some instances there is more or less inequality in the length of the thighs, one femur falling short of the other in its growth. In some instances, as in our specimen, the bones are thin and bent; in some they are thick, even thicker, and with more pronounced ridges, than natural, as though the periosteal tissues not being, as usual, stretched or carried along the bone shaft by the extension resulting from the epiphysal growth at the ends, which growth is deficient in these cases, remain thicker, more vascular, and with more bone-forming capacity than usual upon the shafts. In some instances, where the bones are thus thick as well as short, there is little, or, it may be, no abnormal curvature of the shafts, the only evidences of the rickety condition then being the expressed epiphysal lines and the shortness of the bones, with perhaps this abnormal thickness and some asperities of the shafts. This is the case with a skeleton in the Musée Dupuytren in Paris, in which the flexures are slight, and it is the same in the skeleton at Norwich, of a man, aged 35, described by Mr. Hutchinson in the *Transactions of the Pathological Society*, xl, 229. The latter skeleton I have often seen, and notwithstanding the doubts expressed by that high authority—Mr. Hutchinson—I cannot but regard it as an example of rickets. The bulging of the lower epiphysal lines of the thigh bones, so marked in that skeleton, would alone appear to me sufficient evidence that rickets had in early life been the cause of the dwarfing and other peculiarities in this skeleton.

There are, however, other rickety features in that remarkable skeleton, which is now in the museum of the Norfolk and Norwich Hospital. Not only is there the peculiar bulging of the epiphysal lines at the lower end of the femur, at both ends of the tibia, at the upper end of the fibula, at the upper end of the humerus, and at the lower ends of the radius and ulna; there is also distinct curvature of the fibula backwards with the ridge, usual in rickets, thrown out in front. Both radius and ulna are preternaturally bent backwards, and the like is the case with the humerus in its lower half; the femur is bent a little forwards, the linea aspera is somewhat pronounced, and the neck of the bone has descended so as to form a right angle with the shaft. Moreover, the pelvis presents the antero-posterior flattening often observed in rickets, which is due to flexures in the sides of the iliac bones

above the sacro-sciatic notches, and the sacrum has yielded under the weight on its promontory so as to be turned on its transverse axis, and to have assumed an almost horizontal position. The ribs, from the third to the sixth inclusive, are slightly bent in front, so as to cause some flattening of the chest, and they are remarkably broad. The clavicles are bent upwards, and their external ends are flattened beneath, instead of presenting the usual truncated form. The vertebral column is well developed, and the skull fairly developed, but the limbs have not grown in due proportion to them.

In contrasting the rickety dwarfs with the true dwarfs, I must add that the moral, and perhaps the intellectual, balance appears, on the whole, not to be in favour of the former. Possibly the consciousness of disadvantage resulting from deficiency of stature or from deformity, or the indulgences accorded to such sufferers in childhood, may operate prejudicially upon the moral character, or there may be a closer relation between the development of the intellectual, the moral, and the physical than we are in the habit of recognising. The Norwich skeleton is that of a man who was executed in 1819 for the murder of his child and the attempted murder of his wife, and it is said of him that his personal deformity was in singular unison with his moral depravity. That acute observer and portrayer of men and manners—Charles Dickens—has well exemplified this point in the person and character of Quilp given in the *Old Curiosity Shop*: a large-headed, ugly-faced, bent-legged dwarf, who bullied and whipped his wife, and gloated in all sorts of cruelty, malice, and mischief. It is worthy of remark, as bearing upon the relation of the moral with the physical, that Mr. Roberts, whose observations on anthropometry are well known, finds that lunatics and criminals both show some deficiency in stature and weight as compared with the general population.²

REMARKS

ON

RENAL DROPSY:

A CRITICAL EXAMINATION OF DR. DICKINSON'S THEORY.

By GEORGE JOHNSON, M.D., F.R.S.,

Consulting Physician to King's College Hospital, Physician Extraordinary to Her Majesty the Queen.

DR. DICKINSON, in his learned and eloquent Harveian Oration,¹ has propounded a theory of renal dropsy which appears to me to be inconsistent with some well ascertained facts and acknowledged principles relating to the physiology of the circulation.

The orator refers to the indisputable fact that, in cases of cardiac dropsy, the dropsical effusion is the result of backward pressure through the veins upon the capillaries; and he now maintains that renal dropsy is the result of pressure acting from the heart through the arteries upon the capillaries. He admits the existence of hypertrophy of the left ventricle and of the muscular coat of the arterioles, and accepting this as evidence of an impediment to the onward flow of the blood, he says "the next question is where precisely is the arterial exit impeded, in the arterioles or in the capillaries?.....If the primary obstacle is in the arterioles, as Johnson supposes, the blood must be in a measure shut off from the capillaries, the pressure on them reduced, exudation from them lessened, and the view of renal dropsy which I am about to submit abandoned; but the doctrine of capillary obstruction preponderates, and, to use a political expression, holds the field. It has been urged that the capillaries are not muscular; on the other hand, it has been shown by Roy and

¹ See remarks on this in paper on Growth of Bones, etc., by me in the *Medico-Chirurgical Transactions*, xxliv, 120.

² Report of the Anthropological Committee of the British Association, 1883.

¹ BRITISH MEDICAL JOURNAL, October 24th, 1891.

(Graham-Brown that they are contractile, beside which we cannot doubt, what has long been inferred, that the blood may acquire peculiarities which add difficulties to its transit."

Upon this statement I would remark first, with regard to the blood pressure in the capillaries, that Dr. Dickinson apparently overlooks the fact that the resistance offered by the hypertrophied arterioles is counterbalanced by the increased force of the correspondingly hypertrophied left ventricle, so that the intracapillary pressure probably remains practically unchanged whatever may be the degree of cardiac and arterial hypertrophy.

Then, with regard to contraction of the capillaries, admitting the possibility that, as Roy and Graham-Brown suggest, "the individual capillaries contract or expand in accordance with the requirement of the tissues through which they pass," surely no physiologist maintains that capillary contraction, or any acquired peculiarity of the blood, can cause such an impediment to the circulation as will account for the enormous hypertrophy of the left ventricle, which occurs in cases of chronic Bright's disease.

With respect to the contraction of the capillaries, Dr. Michael Foster remarks:² "In a young animal, at all events, the capillary wall is to a certain extent contractile; the epithelioid cells, which then appear to contain a large amount of undifferentiated protoplasm, seem able, under the influence of stimuli, to change their form, passing from a longer and narrower shape to a shorter and broader one, and thus influencing the calibre of the tube of which they form the walls. How far such an active change of form takes place in the capillaries of the adult body has not yet been definitely determined."

In opposition to Dr. Dickinson's theory, I maintain that the explanation of the hypertrophied left ventricle, which I first published in the fifty-first volume of the *Medico-Chirurgical Transactions*, "holds the field."

When Drs. Alison and John Reid were investigating the phenomena of asphyxia, the hypothesis of capillary obstruction appeared to be consistent with the facts; but since the function of the muscular arterioles, as distinguished from that of the larger elastic arteries, has been revealed by the researches of Bernard, Brown-Séquard and others, the older hypothesis has been shown to be untenable.

It is now admitted by all physiologists that the function of the muscular arterioles is to regulate the blood supply to the capillaries. In the words of Professor Huxley: "While the small arteries lose the function which the capillaries possess of directly irrigating the tissues by transudation, they gain that of regulating the supply of fluid to the irrigators or capillaries themselves. The contraction or dilatation of the arteries which supply a set of capillaries comes to the same result as lowering or raising the sluice gates of a system of irrigation canals."³

The hypertrophy of the muscular arterioles, which I first described in the paper referred to, and the hypertrophy of the left ventricle are joint results of the increased arterial contraction (what I have ventured to call their "stop-cock function") excited by the abnormal condition of the blood in cases of Bright's disease.

Dr. Dickinson admits that in those cases of Bright's disease in which cardiac and arterial hypertrophy with resulting arterial tension are most conspicuous, namely, cases of contracted granular kidney with copious secretion of urine, dropsy is commonly absent, and seeing, as he could not fail to do, that this is inconsistent with his theory of capillary obstruction, he says: "The arterial thickening cannot fail, so far as I see, to obstruct the access of blood to the capillaries and dam it back in the heart and larger vessels with the result of increased tension and increased cardiac hypertrophy. Here comes in the stop-cock action and with it lessened capillary pressure and exudation."

It will be seen, then, that Dr. Dickinson, denying the influence of the arterioles as a cause of the obstruction which results in cardiac hypertrophy, and explaining the occurrence of renal dropsy by increased capillary obstruction and pressure, is driven to admit the obstructive influence of the arterioles in

those cases in which, the cardiac hypertrophy being extreme and dropsy absent, his theory of capillary obstruction is utterly inadequate to explain the facts. How does Dr. Dickinson explain the supposed transfer of the obstruction from the capillaries in one stage or form of disease to the arterioles in another?

It would be interesting to know whether Dr. Dickinson still maintains the view of the function of the arterioles which he has expressed in his work on *Albuminuria*, Ed. ii, p. 328, where he says "the only feasible view of the mechanism of the circulation is that the wave of blood which arises in the ventricle is helped on by an advancing zone of arterial contraction, which follows swiftly in its rear, and by a sort of vermicular action continues the ventricular impulse as far as arterial muscle extends."

He dismisses the stop-cock theory as improbable and implying an unnatural conflict between the heart and the arteries. He further states: "We revert to the older view of capillary hindrance, and in this, it is scarcely possible to doubt, we find the key of the whole mystery of cardio-vascular hypertrophy."

It is evident that at the time of this publication Dr. Dickinson believed that the hypertrophy of the arterioles is the result of an effort to assist the heart in overcoming an obstruction caused by the capillaries in front.

In my *Medical Lectures and Essays*, page 38, I have suggested the following explanation of renal dropsy: "When, in consequence of acute inflammatory disease of the kidney, the urinary crystalloids and water accumulate in the blood, they pass thence by an exosmotic process through the walls of the capillaries into the subcutaneous areolar tissue and into one or more of the serous cavities. On the other hand, when the kidney has reverted to its normal condition and recovered its functional activity, the blood being first freed from excess of urinary solids and water, a current of dissolved urinary crystalloids sets in from the tissues into the blood and out by the kidneys. Thus, by a copious flow of urine the dropsical accumulation is soon swept away."

These phenomena, like the increased secretion of diabetic urine, the result of an excess of a saccharine crystalloid in the blood, occur, as I believe, quite apart from the influence or the existence of increased capillary pressure.

AN ADDRESS

ON THE

SURGERY OF THE TONGUE.

Read before the Liverpool Medical Institution.

By JONATHAN HUTCHINSON, F.R.C.S., F.R.S., LL.D.,
Late President of the Royal College of Surgeons.

THE surgery of the tongue has been greatly developed within the last five-and-twenty years. I well recollect the time when operations on the tongue were regarded with the utmost dread on account of the fear of hæmorrhage. Removal of the entire organ was never thought of at the London hospitals during the early part of my familiarity with their operating theatres. Even partial operations were rare, and, although so infrequent, I remember witnessing several in which the patient almost died on the operating table. They were never undertaken excepting in cases in which the disease was so far advanced that the diagnosis of cancer was obvious to everyone; and it is needless to add that they were seldom attended by results which offered encouragement to the performance of an operation in itself attended by so much hazard. The return of the disease in the glands was always a matter of certainty and promptitude.

I shall never forget the first case in which I was induced to operate on the tongue. This was in 1858, and about two-thirds of the organ were removed. The case was one of obvious cancer in a man of middle age. I operated in a lodging at Reigate, where I had induced the man to go, partly because I had myself a house there, and partly that he might have the benefit of pure air. Such was my fear of bleeding that I had had

² *Journal of Physiology*, vol. 2, p. 358.

³ *Textbook of Physiology*, Ed. v, p. 192.

⁴ *Mental Physiology*, 1886, p. 25.